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Original Investigation

A social contextual analysis of youth cigarette smoking development

Susan T. Ennett, Ph.D.,[1] *Vangie A. Foshee, Ph.D.,*[1] *Karl E. Bauman, Ph.D.,*[1] *Andrea Hussong, Ph.D.,*[2] *Robert Faris, Ph.D.,*[3] *John R. Hipp, Ph.D.,*[4] *& Li Cai, Ph.D.*[5]

[1] *Department of Health Behavior and Health Education, University of North Carolina at Chapel Hill, Chapel Hill, NC*
[2] *Department of Psychology, University of North Carolina at Chapel Hill, Chapel Hill, NC*
[3] *Department of Sociology, University of California at Davis, Davis, CA*
[4] *Department of Criminology, Law, and Society, University of California at Irvine, Irvine, CA*
[5] *Division of Social Research Methodology, University of California at Los Angeles*

Corresponding Author: Susan T. Ennett, Ph.D., *Department of Health Behavior and Health Education, University of North Carolina at Chapel Hill, Campus Box 7440, Chapel Hill, NC 27599, USA. Telephone: 919-966-9207; Fax: 919-966-2921; E-mail: sennett@email.unc.edu*

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Abstract

Introduction: We apply a social contextual perspective based on Bronfenbrenner's ecology of human development theory to understanding development of youth cigarette smoking. We examine the contributions of family, peer, school, and neighborhood contexts. Context attributes examined were derived from social learning and social control theories.

Methods: Data are from 6,544 youth who participated in at least one of five waves of data collection between Spring 2002 and Spring 2004, 1,663 randomly selected parents who participated in one or more of three waves of data collection in the same time period; and the U.S. Census. Three-level hierarchical growth models were used to examine the contributions of time-varying measures of the four social contexts to development of smoking from age 11–17 years. Interactions between variables were examined within and between social contexts.

Results: Attributes of each social context made independent contributions to adolescent smoking development; there also were significant interactions between variables from different contexts indicating joint contextual effects. Attributes of the social bond moderated exposure to models of smoking within and between the family and peer contexts.

Discussion: Results suggest the value of a social contextual perspective in research on the etiology of youth smoking development as well as the utility of guidance by social learning and social control theories. While all contexts were implicated in adolescent smoking, the family and peer contexts were primarily implicated, with findings suggesting the need for consideration of interactive effects between social learning and social control variables within and between these contexts.

Introduction

A social contextual perspective on development of youth cigarette smoking presumes that the multiple social contexts in which young people live influence smoking (Cook, 2003; Flay Petraitis, & Hu, 1999; Wen, Van Duker, & Olson, 2009; Wilcox, 2003). Such a perspective also presumes that factors emanating from any one social context are best understood considering factors present in other social contexts. That is because adolescents inhabit numerous layered social contexts: they are nested within families and friendships that are nested within neighborhoods and schools nested within communities nested within states and countries. The embeddedness of youth's social contexts suggests the possibilities of redundant, amplifying, countervailing, and unique social forces on youth smoking. Because of the interrelationships among youth social contexts, focus on only one context risks overestimating its contributions to adolescent smoking. It also risks attributing effects to one context that may be confounded with another. Furthermore, focus on single contexts misses how contexts might jointly impact adolescent smoking (Cook). Our purpose is to examine the concurrent and joint contributions to adolescent smoking of their key social contexts of families, peer groups, schools, and neighborhoods. We examine development of smoking from early to mid-adolescence, with smoking measured along a continuum from none to emerging dependence.

Given the conceptual and analytic complexities inherent in simultaneously examining several social contexts, an essential consideration is guidance by theory on what about the social contexts to study and how to conceptualize relationships among social factors and social contexts (Cook, 2003; Flay et al., 1999; Ennett, Foshee, et al., 2008). Selection among the myriad of contextual factors, much less hypothesis generation and interpretation of findings, is impossible without theoretical guidance. We draw on three theories—Bronfenbrenner's ecology of human development (Bronfenbrenner, 1977), social learning theory (Akers, Krohn, Lanza-Kaduce, & Radosevich, 1979; Bandura, 1977; Petraitis, Flay, & Miller, 1995), and social control theory (Elliott, Huizinga, & Ageton, 1985; Hirschi, 1969; Petraitis et al.). The latter two theories point, respectively, to study of the presence of smokers in each of the social contexts who may serve as role models of the behavior and to examination of attributes of the social bonds within each context that could

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constrain or encourage youth smoking. The ecology of human development suggests examining interactions between smoking modeling and bond attributes both within and between social contexts.

Theoretical framework

Bronfenbrenner's ecology of human development (Bronfenbrenner, 1977) provides the overarching conceptual framework. The central precepts are that human development takes place within a set of nested and changing environments and that interactions within and between those environments shape behaviors. Most fundamentally, the ecology of human development suggests the contexts to study. The family, peer, and school contexts are identified as microsystems and the neighborhood as an exosystem. Microsystems are defined as the proximal and major settings in which youth development takes place; exosystems are more distal social environments within which microsystems are situated. Microsystems are expected to have primacy over exosystems in influencing development. Both microsystems and exosystems are assumed to be multifaceted in how they influence development.

In describing how to conceptualize interrelationships among these contexts, Bronfenbrenner indicates that the most important effects are likely to be interactions. He constructs mesosystems as the interrelations among microsystems and thus among the family, peer group, and school. The concept of a mesosystem exemplifies the interdependency among contexts that is central to the theory and suggests operationalization by examination of multiple factors describing microsystems and interactions of such factors between microsystems. In contrast to the interdependency in mesosystems, Bronfenbrenner suggests that exosystems tend to impinge on microsystems and mesosystems in a unidirectional rather than bidirectional fashion. Thus, interactions between the neighborhood as an exosystem and the microsystems of family, peer group, and school are not suggested.

While describing the multifaceted nature of social contexts and the interrelations between contexts, Bronfenbrenner's ecology of human development does not identify specific contextual attributes or processes to investigate. We turn here to social learning (Akers et al., 1979; Bandura 1977; Petraitis et al., 1995) and social control (Elliott et al., 1985; Hirschi, 1969; Petraitis et al.) theories because they situate causes of youth smoking in the social environment, and they are the two most supported theories in research on adolescent smoking.

Social learning theory emphasizes the facilitating effect on youth smoking of exposure to smokers who serve as role models, whereas social control theory focuses on the constraining effects on smoking of conventional social relationships. Social learning theory posits that adolescent smoking is learned behavior acquired through social interactions and reinforcement (Petraitis et al., 1995). The widely documented relationships between adolescent smoking and their friends' (e.g., Kobus, 2003), siblings' (e.g., Bricker et al., 2006) and, less consistently, parents' smoking (e.g., Avenevoli & Merikangas, 2003) can be seen as evidence of social learning. Similarly, although less often documented, youth smoking has been associated with exposure to smoking models among schoolmates (Bricker, Andersen, Rajan, Sarason, & Peterson, 2007; Ellickson, Bird, Orlando, Klein, & McCaffrey, 2003; Leatherdale, Cameron, Brown, & McDonald, 2005).

Social control theory posits that a tendency toward deviance is universally shared but manifested only in the face of weakened conventional controls that reside in families and other social groups (Petraitis et al., 1995). Those controls may be attenuated by strained relationships, such as in high-conflict families (Duncan, Duncan, Biglan, & Ary, 1998; Flay, Hu, & Richardson, 1998). In contrast, conventional bonds exemplified by parental monitoring and supervision (e.g., Biglan, Duncan, Ary, & Smolkowski, 1995; Hill, Hawkins, Catalano, Abbott, & Guo, 2005) and supportive parent–child relationships (e.g., Doherty & Allen, 1994; Scal, Ireland, & Borowsky, 2003) deter youth smoking. Such relationships can be understood from a social control perspective as evidence for the constraining influence of conventional parent–child bonds. Similarly, inverse relationships between youth substance use and school connectedness (Battistich & Hom, 1997; Henry, Stanley, Edwards, Harkabus, & Chapin, 2009) provide evidence for the protective effect of conventional school bonds. Although focused on violence and delinquency rather than on smoking, neighborhood features such as social cohesion and informal social control have been conceptualized within the context of social control theory as deterrents to youth misbehavior (e.g., Brook, Nomura, & Cohen, 1989; Sampson, Raudenbush, & Earls, 1997).

Conceptual framework and study hypotheses

We simultaneously examine attributes of adolescents' family, peer, school, and neighborhood social contexts that could influence their smoking. Based on etiological evidence in support of social learning theory, we focus on social modeling of smoking. From social control theory, we focus on three attributes of social bonds that could constrain or facilitate smoking—closeness, social regulation, and strain. Following from Bronfenbrenner's conceptualization of interrelationships, we examine interactions between social modeling and the three social bond attributes within the four social contexts and between the three microsystems (family, peers, and school) that interact to form mesosystems (see Figure 1).

Interactions between social learning and social control-related variables have received limited attention in prior studies of youth smoking, with inconsistent evidence as to whether attributes of the social bond moderate effects of modeled smoking behavior (Bricker et al., 2009; Den Exter Blokland, Hale, Meeus, & Engels, 2007; Doherty & Allen, 1994; Chassin et al., 2005; Urberg, Luo, Pilgrim, & Degirmencioglu, 2003; White, Johnson, & Buyske, 2000; Wilson, McClish, Heckman, Obando, & Dahman, 2007). When such interactions have been studied, interactions within contexts—typically, the family—rather than between contexts have generally been the focus (Foshee & Bauman, 1992; Simons-Morton, 2002). Our study contributes to understanding of social processes involved in youth smoking by applying Bronfenbrenner's insistence on the primacy of interactions to examination of whether attributes of the social bond moderate exposure to smoking roles models within (microsystems and exosystem) and between social contexts (mesosystems).

With one exception, our expectations regarding the nature of interactions between the social learning and social control variables vary depending on the contexts involved. Within the family and neighborhood contexts, where adult norms against smoking are expected, we hypothesize that closeness and social

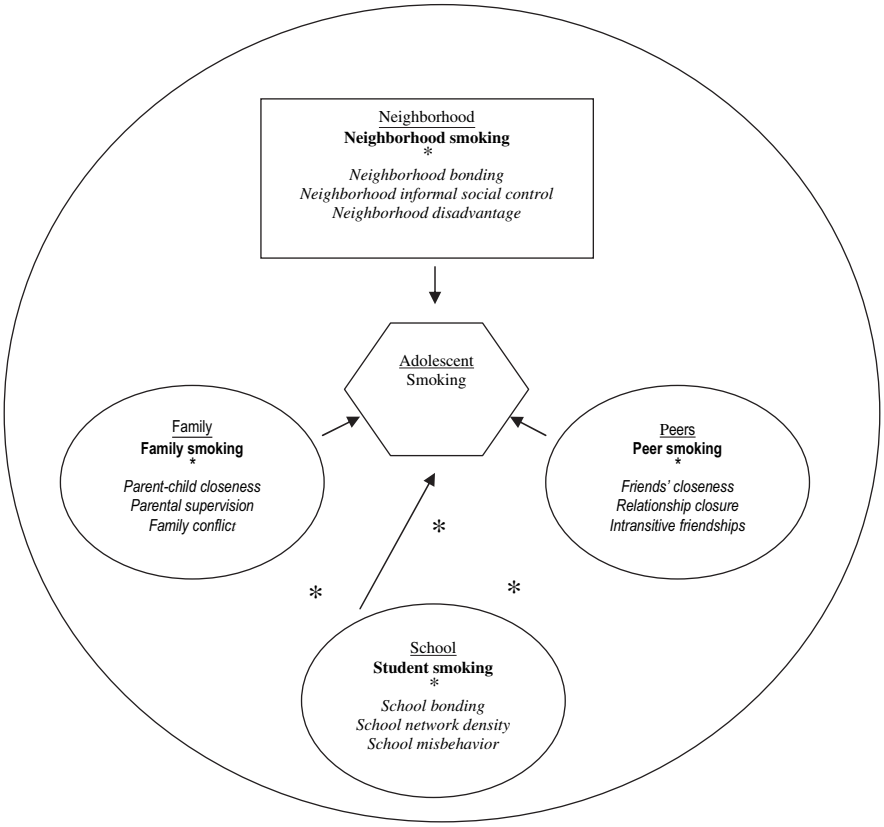

Figure 1. Conceptual framework of the social context of adolescent smoking based on the ecology of human development, social learning, and social control theories. Family, peer, school, and neighborhood contexts and their relationships to each other are suggested by the ecology of human development; ovals depict microsystems, *'s depict interactions between microsystems (mesosystems) and between social learning and social control characteristics within contexts, and the rectangle depicts an exosystem. Characteristics of contexts in bold are derived from social learning theory (modeling smoking), and characteristics in italics are derived from social control theory (closeness, social regulation, and strain).

regulation will buffer effects of family and neighbors' smoking. Within the peer and school contexts, where the reference is to interactions with other adolescents, closeness and social regulation are hypothesized to amplify effects of friends' and schoolmates' smoking. Consistent with these hypotheses, we expect that between-context interactions involving family closeness and social regulation will buffer exposure to smoking by friends and schoolmates, while peer closeness and social regulation will amplify exposure to smoking by schoolmates. In the exception, we expect that strain will consistently magnify the effect of smoking by others within and between all contexts.

The fact that we are examining four social contexts introduces complexities in how attributes are measured and in expectations about the nature of the interactions between the social learning and social control variables. Regarding measurement, following from Bronfenbrenner, we take a systems approach to measuring families, friendship groups, neighborhoods, and schools at the contextual or aggregate level. We use social network analysis to measure selected attributes of peer groups and schools, and we use data collected from random samples of residents to measure neighborhood characteristics. In operationalizing constructs, smoking modeling is measured in the same way across contexts by the presence of smokers, but the three social bond measures are tailored to each construct, as shown in Figure 1.

Significance

Researchers have called for more comprehensive social contextual studies of the etiology of youth smoking dating back over the last decade (Cook, 2003; Flay & Clayton, 2003; Flay et al., 1999; Kaufman & Feiden, 1999). Most studies, however, remain focused on the peer and/or family contexts, with few examples of multicontext studies (Wen et al., 2009). Researchers also have noted the need to study the development of smoking over time and the emergence of dependence (Conklin, Clayton, Tiffany, & Shiffman, 2004; Flay & Clayton, 2003). We address these gaps in the current study.

Methods

Study overview

Data are from a longitudinal study of contextual factors that influence adolescent smoking and other problem behaviors. The study design included four components to enable contextual analyses: (a) a census of adolescents identified by school enrollment and surveyed in school every 6 months for a total of five assessments (Waves 1–5), (b) a simple random sample of parents of the adolescents surveyed annually by telephone for a total of three assessments (Waves 1, 3, and 5), (c) social network analysis of school networks based on friendship nominations collected in the school surveys, and (d) geocoding of adolescent

addresses to allow linkage to U.S. Census block groups. Data collection with adolescents and parents began in Spring 2002 and ended in Spring 2004.

All protocols were reviewed and approved by the Institutional Review Board at the University of North Carolina at Chapel Hill. The board approved a waiver of written parental consent, although parents could refuse their child's participation by returning a postage-paid form or by calling a toll-free number. Written adolescent assent was obtained at the time of data collection.

Adolescent sample and data collection

Adolescents in middle and high school grades in three complete public school systems in North Carolina participated. The school systems included a total of eight middle schools, two K-8 schools, six high schools, and three alternative schools with middle and high school grades. Adolescents were in the 6th, 7th, and 8th grades at Wave 1 and in the 8th, 9th, and 10th grades at Wave 5. At each assessment, all enrolled students at the targeted grade levels, except for those in self-contained classrooms for exceptional children and those with limited English comprehension, were eligible. Thus, new students at each school joined the study at each wave of data collection, with almost one quarter of the sample enrolling after the first assessment wave. Across the five assessments, the samples ranged in size from 5,220 (Wave 1) to 5,017 (Wave 5) with 6,891 unique cases across all waves. Response rates at the five waves were 88.4%, 81.3%, 80.9%, 79.1%, and 76.0%, respectively.

Parent sample and data collection

A simple random sample of 1,663 parents of adolescents who completed the Wave 1 survey completed a 25-min telephone interview at Waves 1, 3, and 5. By design, in the majority of cases (98.2%), the mother/female caretaker was the parent interviewed. Response rates at the three waves were 79.8%, 82.5%, and 71.8%, respectively. The parent sample was designed to provide a sufficient number of cases within each Census block group to allow aggregate measures of neighborhood context from parent responses to questions about their neighborhood.

Social network analysis

Social network analysis was conducted on friendships reported by adolescents at each assessment (Ennett, Bauman, et al., 2006). Data collectors gave each student a student directory that alphabetically listed all enrolled students along with a unique four-digit peer identification number. Adolescents identified up to their five closest friends, starting with their best friend. Friends not in the directory were identified by "0000." Because most adolescent friendships are with adolescents in the same school and grade, we bounded social networks by school and grade, with the following exceptions. In high schools and alternative schools, networks were bounded by school only because classes and activities were not grade segregated, and therefore, cross-grade friendships were likely. In the two K-8 schools, networks also were bounded by school because of their small enrollments. We defined the peer context by the adolescent's set of close friends, including those nominated by the adolescent and those who nominated the adolescent as a friend. The school context was defined by the school network to which the adolescent belonged.

Geocoding

Adolescent addresses were sent to a commercial geocoding firm to be matched with U.S. Census block group data in the 2000 Dicennial Census (U.S. Census Bureau, 2001). At Wave 1, 94.8% of adolescents were matched to a street address, 3.9% to the ZIP centroid, and less than 1% were not matched. The match rates were similar at subsequent assessments. The geocodes represented 158 Census block groups in the three county area as well as some block groups in contiguous areas where some families lived. On average, there were 23.7 block groups per school. We defined the neighborhood context by the U.S. Census block group in which the adolescent lived the first time the adolescent was assessed.

Analysis sample

The analysis sample included all adolescents who participated at any wave of data collection ($N = 6,891$) except for those missing birth date or outside the typical age range of 11 through 17 years for the grades studied ($n = 51$, 0.7%) or who could not be geocoded ($n = 296$, 4.3%), yielding a sample size of 6,544 (95.0%).

The mean self-reported age of adolescents at Wave 1 was 13.12 years ($SD = 1.04$). About half were male (51%), and the self-reported race/ethnicity distribution was 52% White, 37% Black, 4% Hispanic, and 7% other race/ethnicity. Averaged across all five waves of assessment, approximately 13% of adolescents reported living in other than a two-parent family, and for 39%, the highest education attained by either parent was reported by the adolescent to be high school or less.

Measures

Smoking

We measured smoking on a continuum from none to the emergence of dependence as appropriate for examining development of smoking over a several year age span. We constructed a scale measuring recent (past 3 months) smoking using six items from the revised Fagerström Test for Nicotine Dependence (Heatherton, Kozlowski, Frecker, & Fagerström, 1991). The items measured the number of cigarettes smoked daily and indicators of dependence (e.g., difficulty keeping from smoking in forbidden places); except for the number of cigarettes smoked daily, the response options were dichotomous. Even though some adolescents progressed to dependence, the distributions of responses was limited and skewed, as is typical in studies of smoking in general populations of adolescents. We used item response theory (IRT) to construct the scale (Thissen, Nelson, Rosa, & McLeod, 2001) because IRT is a method for scaling responses on multiple categorical indicators to describe an underlying latent construct, which results in a linear latent variable scale. We used the item parameter estimates from a two-parameter logistic IRT analysis, obtained using MULTILOG software (Thissen, Chen, & Bock, 2003), to compute scale scores from the maximum a posteriori method (Thissen & Orlando, 2001). The metric of the resulting scores was a standard normal.

Social context

We measured indicators of smoking modeling, closeness, social regulation, and strain in each social context as described in Table 1. The latter three measures were tailored to each context. Most measures were means of reduced sets of items from existing scales identified through earlier psychometric analysis of

Table 1. Social context measures

Social context attribute	Variable	Number items and source	Sample item	Description	Alpha	Source
Family modeling	Number of current smokers in family (mother, father, and siblings)	Three from adolescent report	About how many cigarettes do you think she (mother) now smokes in a day?	Count (range 0–6)	—	—
Family closeness	Parent–adolescent closeness	Six from adolescent report	How close do you feel toward her (mother)?	Mean (range 1–4)	.80	—
Family social regulation	Parental supervision of adolescent	Six from adolescent report	She (mother) has rules that I must follow	Mean (range 1–4)	.84	Jackson, Henriksen, and Foshee (1998)
Family strain	Family conflict	Three from adolescent report	We fight a lot in our family.	Mean (range 1–5)	.85	Bloom (1985)
Peer modeling	Smoking of friends	Six per friend from friend report	—	Mean	—	Heatherton et al. (1991)
Peer closeness	Mutual friends' closeness	One per reciprocated friend from friend report	How close do you feel toward (best friend)?	Mean (range 1–4)	—	—
Peer social regulation	Relationship closure	Three per friend from adolescent report	Have you met your friend's parents?	Mean (range 0–1)		Bearman and (2004)
Peer strain	Membership in intransitive triads	From social network analysis	—	Proportion of intransitive triads among all triads to which the adolescent belonged	—	Moody (2000, 2001)
School modeling	Smoking of schoolmates	Six per student from student report	—	School network mean	—	Heatherton et al. (1991)
School closeness	School bonding	Three per student from student report	Students in this school treat each other with respect	School network mean (range 1–5)	.84	Battistich and Hom (1997)
School social regulation	School relative density	From social network analysis	—	Proportion of friendship ties present among all possible ties, adjusted for the fixed number of friend nominations		Moody (2000, 2001)
School strain	School misbehavior	Five per student from student report	During the past 3 months, how many times have you gone to school but skipped classes?	School network mean	—	Farrell, King, White, and Valois (2000)
Neighborhood modeling	Smoking of adolescents in neighborhood	Six per adolescent resident from resident report	—	Block group mean	—	Heatherton et al. (1991)
Neighborhood closeness	Neighborhood bonding and trust	Three per parent resident from parent report	People in this neighborhood can be trusted.	Block group level factor scores obtained from confirmatory factor analysis	.90	Parker et al. (2001)
Neighborhood social regulation	Neighborhood informal social control	Six per parent resident from parent report	. . . how likely or unlikely is it your neighbors would step in and do something . . . if teens were damaging property?	Block group level factor scores obtained from principal-components analysis	.91	Sampson et al. (1997)
Neighborhood strain	Neighborhood disadvantage	Six per parent resident from parent report and five per block group from Census data	There is a lot of crime in your neighborhood.	Block group level factor scores obtained from confirmatory factor analysis	.84	Ross and Jang (2000); U.S. Census Bureau (2001)

data collected on full scales in a pilot study. All measures were constructed to be time varying. Measures of the peer, school, and neighborhood contexts were all constructed as means or proportions to account for varying sizes of the contexts. We provide elaboration here for three social network-based measures whose meanings may not be apparent.

Relationship closure, the indicator of peer social regulation, was the mean of three items per nominated friend measuring whether the adolescent's parents had met the friend, the adolescent had met the friend's parents, and adolescent and friend's parents had met (Bearman & Moody, 2004). Higher values indicate greater parental knowledge of friends, suggesting greater intimacy between adolescents and perhaps more effective oversight by parents (Coleman, 1988). Peer strain was measured by the proportion of intransitive triads to which adolescents belonged, where an intransitive triad is a set of three peers linked through friendship nominations such that a friend's friend is not a friend. Whereas transitive triads represented balanced closed friendship circles, intransitive triads reflected unbalanced and potentially discordant relationships where an adolescent's friendships were not overlapping and thus carried the potential for strain. In support of this possibility, Bearman and Moody demonstrated that adolescent females with higher proportions of intransitive relationships were at risk for suicide ideation; they argued that intransitive friendships lead to competing normative pressures that lower effective normative regulation.

School network relative density, the measure of school social regulation, was the proportion of possible friendship ties present in the school network, adjusted for the fixed number of friend nominations (Moody, 2000, 2001). Higher values indicated greater presence of friendships linking together adolescents in the school and thus more closely knit school communities with presumed greater potential for regulating behavioral norms.

Demographics

Measures were based on adolescent self-report. Age was measured continuously based on dates of birth and assessment. Sex was coded, so the reference group was female. Race/ethnicity was based on the adolescent's modal response across all assessment waves and dummy coded to include White (reference group), Black, Hispanic, and other race/ethnicity. Family structure was coded as two parents in the home (reference group) versus some other composition. Parent education measured the highest education attained by either parent and coded as high school graduate or less versus more than high school graduate (reference group). Because family structure and parent education could change over the course of the study, the two variables were coded as the average across the five waves.

Multiple imputation

To provide complete data sequences for all cases (Allison, 2002), we used PAN, a multilevel multiple imputation program appropriate for longitudinal data, to impute five sets of values for any missing data from adolescents or parents (Schaefer, 2001). Adolescent data were missing primarily due to respondents not completing all five assessments because of either being lost to follow-up or, as allowed by the study protocol, not entering the study until Waves 2–5. Of the 6,544 students in the analysis sample, 44.2% participated in all the five waves, 18.3% participated in four waves, 15.2% in three waves, 10.9% in two waves, and 11.5% at only one wave. Adolescents missing at one or more waves compared with those continuously assessed once they entered the study were significantly more likely to be male, Black or of other race/ethnicity, live in other than a two-parent household, have parents with lower education, and have higher levels of smoking. All these demographic variables are controlled in analysis.

Of the 1,663 parents, all completed the first wave of data collection; 66.8% completed all three interviews, 20.4% completed either the first and second or the first and third interviews, and 12.8% completed only the first interview. Parents who completed one or two interviews compared with those who completed all three interviews were more likely to be Black, live in other than a two-parent household, and have lower education.

Statistical analysis

Because of the nestedness of our data, such that repeated measures of smoking were nested within adolescents and adolescents were nested within neighborhoods and schools, we used a multilevel modeling approach. Specifically, we estimated three-level hierarchical growth models with time specified at level one, adolescents at level two, and neighborhood at level three. We specified neighborhood rather than school at level three because of the larger number of neighborhoods than schools and because neighborhoods were nested within schools.

The data were arranged in a cohort sequential design whereby data collected over approximately two and one half years from the three grade cohorts were merged to allow accelerated growth curves of smoking to be modeled over approximately 6 years. We used age to measure time to allow change in smoking to be modeled from age 11 through age 17 years (Mehta & West, 2000). We established the appropriateness of the cohort sequential design by determining that the cohorts did not differ in smoking growth curves. We demonstrated the lack of cohort differences by a likelihood ratio test comparing the unconditional model (described below) with a model that added a variable measuring cohort and the interaction between cohort and age; the test was not significant (Miyazaki & Raudenbush, 2000).

We report the analysis in stages beginning with estimation of the unconditional model to determine the random components and form of the smoking growth curve, with an a priori expectation of a linear model. We next estimated a series of conditional models, beginning with two preliminary models, followed by five primary models. The first preliminary model included only demographic variables; all subsequent models controlled for these variables. The second preliminary model included all four sets of variables describing the family, peer, school, and neighborhood contexts but did not include any interactions among variables within or between contexts. For this model only, we computed standardized coefficients representing the *SD* change in smoking expected from a 1 *SD* change in the predictor variable to allow comparison of the size of variable effects across contexts. We did not compute these standardized coefficients in the primary models because of interpretational difficulties in the presence of interactions.

The five primary conditional models built on each other. The first included the variables and their interactions characterizing

the family, peer, and school contexts (microsystems). The second added the set of variables and their interactions characterizing the neighborhood (exosystem). The final three models added between-context interactions involving family and peers, family and school, and peers and school (mesosystems). In addition to the hypothesized interactions between smoking modeling and social bond variables, for all three mesosystem models, we also included the between-context interaction between the two indicators of smoking modeling because of the possible risk associated with accumulated exposure (Bricker et al., 2006).

For each conditional model, we report the coefficients for the fixed effects of the variables and, if included in the model, their interactions. We also report the *F* statistic for testing the significance of the set of variables and interactions added to each successive model. Because the social context variables were time varying, a significant effect means that the relationship between the social context variable (or interaction between context variables) and adolescent smoking was significant on average over the ages examined. For ease of interpretation, we refer throughout to each contextual attribute by the construct name (e.g., peer strain) rather than the specific indicator (e.g., intransitive friendships triads).

All analyses were conducted using SAS v. 9.1.3., using PROC MIXED and PROC MIANALYZE (SAS, 2002–2003).

Results

Unconditional model

The best-fitting unconditional model was a linear growth model with random individual and neighborhood intercepts and slopes. The model demonstrated significant individual (Z-score = 5.54, $p < .001$) and between-neighborhood (Z-score = 3.14, $p < .01$) variation around the mean intercept centered at age 12 years and significant individual (Z-score = 5.08, $p < .001$) and between-neighborhood (Z-score = 3.48, $p < .001$) variation around the slope. The model also showed significant fixed effects such that the mean intercept for smoking was significantly different from zero ($B = -.02$, $SE = 0.01$, $p < .05$), and there was significant growth in smoking through age 17 years ($B = .08$, $SE = 0.004$, $p < .001$). The linear model was a better fit than a quadratic model; the spline model did not converge.

Preliminary conditional models

Demographics model

In the preliminary model including only the demographic variables, both age ($B = .07$, $SE = 0.01$, $p < .001$) and high school enrollment ($B = .03$, $SE = 0.01$, $p < .05$) significantly predicted increased smoking. Because the remaining demographics were time invariant, their relationships with both the intercept and slope of the growth curves were modeled. Black and Hispanic youth smoked at the same initial levels as White youth, but adolescents of other race/ethnicities had higher initial levels of smoking compared with White youth ($B = .09$, $SE = 0.03$, $p < .001$), as did those not in two-parent families ($B = .04$, $SE = 0.02$, $p < .05$) and whose parents were high school graduates or less versus those with more than a high school education ($B = .06$, $SE = 0.02$, $p < .001$). Black ($B = -.05$, $SE = 0.01$, $p < .001$) and Hispanic youth ($B = -.04$, $SE = 0.01$, $p < .01$) increased their smoking at slower rates than white youth. Adolescents in other family compositions than two-parent families ($B = .03$, $SE = 0.01$, $p < .01$) and whose parents had a high school education or less ($B = .03$, $SE = 0.01$, $p < .01$) increased their smoking at higher rates than their counterparts.

Main effects model

All family variables significantly predicted adolescent smoking in the expected direction, as did all peer variables except peer closeness, which was not a significant predictor (Table 2). In the school and neighborhood contexts, only modeling of smoking significantly predicted increased adolescent smoking. Within each context, the largest *SD* change in smoking was associated with the modeling effect. In the family context, which measured the number of smokers in the family including siblings, every additional smoker in the family resulted in a 0.12 *SD* change in adolescent smoking. In the peer, school, and neighborhood contexts, where modeling was measured as the mean smoking value among the context members, a 1 *SD* increase led to a 0.11, 0.04, and 0.15, respectively, *SD* change in adolescent smoking. To provide perspective, in the demographic model presented above, one unit changes in age and parent education led to 0.17 and 0.04 *SD* increases in adolescent smoking and the smoking intercept, respectively (results not shown). With the exception of family strain (standardized smoking beta = .10), the standardized coefficients for change in smoking associated with the

Table 2. Main effects of social context predictors on smoking from age 11 through age 17 years (*N* = 6,544)

	Smoking		
	B (*SE*)	*SD*	Standardized *B*[a]
Family context			
Modeling	0.08 (0.00)***	0.87	0.12
Closeness	−0.04 (0.01)***	0.63	−0.05
Social regulation	−0.03 (0.00)***	0.80	−0.05
Strain	0.04 (0.00)***	1.14	0.10
Peer context			
Modeling	0.17 (0.01)***	0.33	0.11
Closeness	−0.00 (0.00)	1.46	−0.01
Social regulation	0.02 (0.00)***	1.04	0.03
Strain	0.10 (0.03)*	0.17	0.03
School context			
Modeling	0.17 (0.05)**	0.11	0.04
Closeness	−0.01 (0.01)	0.33	0.00
Social regulation	0.03 (0.04)	0.12	0.01
Strain	−0.05 (0.04)	0.12	−0.01
Neighborhood context			
Modeling	0.63 (0.03)***	0.12	0.15
Closeness	−0.02 (0.02)	0.23	−0.01
Social regulation	0.01 (0.02)	0.23	0.00
Strain	−0.02 (0.04)	0.11	0.00

Note. Adolescents are nested in neighborhoods. The hierarchical growth model is computed on five imputed datasets and controls for gender, race/ethnicity, family structure, parent education, and high school enrollment.

[a]*SD* change in smoking associated with a 1 *SD* change in the predictor variable.

*$p < .05$; **$p < .01$; ***$p < .001$.

significant social bond variables in the family, and peer contexts were approximately one third to one half the size of the modeling coefficients.

Primary conditional models

Microsystem model

The contributions of the family, peer, and school context variables and their within-context interactions to smoking are shown in Table 3 (microsystem). All the family context variables and the two-way interactions between family smoking and family social bond indicators were significantly associated as expected with smoking from ages 11 through 17 years. Both family closeness and social regulation buffered the detrimental effect of family smoking on adolescent smoking, while family strain magnified the effect.

In the peer context, peer strain increased youth smoking, as expected, but did not magnify the effect of friends' modeling of smoking. As expected, both peer closeness and social regulation amplified the positive relationship between youth smoking and friends' modeling of smoking.

In the school context, only classmates' modeling of smoking positively predicted adolescent smoking, while none of the three social bond variables either moderated the modeling effect or had main effects on youth smoking.

Exosystem model

The addition of the set of neighborhood variables did not make a significant contribution to the model (Table 3, exosystem). Nevertheless, neighbors' modeling of smoking was positively associated with the youth smoking trajectories. None of the neighborhood social bond variables predicted smoking or moderated the effect of neighbors' smoking.

With the addition of the neighborhood variables, all the significant relationships between adolescent smoking and the family, peer, and school context variables remained unchanged, although some significance levels were attenuated. The interaction between social regulation and smoking modeling in the school context, however, became significant, such that the modeling effect was decreased when social regulation was greater rather than lower. The isolated nature of this change suggests the possibility that it was due to chance.

Mesosystem models

The cross-context interactions included in Family × Peers and Family × School mesosystem models (Table 3) were each significant additions to the model. In both these mesosystem models, family strain and family smoking magnified the effects of friends' and schoolmates' smoking on youth smoking, while neither family closeness nor family social regulation moderated the effects of smoking models. The interactions included in the Peers × School mesosystem model did not significantly contribute to the exosystem model.

Discussion

In this examination of the social context of adolescent smoking development from ages 11 through 17 years, we found independent contributions from all four key social contexts of the family, peers, school, and neighborhood as well as significant interactions between variables from different contexts indicating joint contextual effects. In addition, we showed that specific contextual attributes derived from social learning and social control theories predicted adolescent smoking development and that there were significant interactions between these variables, such that attributes of the social bond moderated exposure to models of smoking within and between some social contexts. A strength of our analysis was using a measure of smoking that allowed for consideration of increased smoking and the emergence of dependence over a several year age span in adolescence.

Our findings confirm expectations, with some qualifications as elaborated below, motivated by Bronfenbrenner's theory of the ecology of human development. In guiding understanding of how behaviors, such as smoking develop in youth, the theory calls for inclusive consideration of nested social contexts and places emphasis on interactions or joint effects. Our results suggest the value of such a social contextual perspective in research on the etiology of youth smoking as well as the utility of guidance by social learning and social control theories. At the same time, our preliminary main effects model where we used standardized coefficients to allow comparison of variable effect sizes across contexts showed generally small effects, although they were in the range of the effects of age and parent education, two well-established predictors of youth smoking. The small effect sizes are not surprising given the number of contexts examined, that every contextual effect was modeled net of all other contextual effects and demographic controls, that most variables did not share the same measurement source, and that prediction was over a 6-year period. Nevertheless, we caution against overinterpretation of our findings.

We first discuss our findings relevant to the general contextual relationships expected from Bronfenbrenner's theory, then in light of our hypotheses about the specific nature of the interactions between the social learning and social control variables within and between contexts.

Support for ecology of human development relationships

As expected based on the ecology of human development, when considered simultaneously, social processes in the family, peer, and school contexts, all proximal microsystems, and in the neighborhood, a more distal exosystem, contributed uniquely to development of youth smoking between ages 11 and 17 years, net of each other and after accounting for the contributions of demographic characteristics. Increased exposure to smoking models in each context significantly contributed to increased adolescent smoking. Contrary to the primacy of microsystems over exosystems in influencing development, however, the modeling effect was stronger in the neighborhood than in the peer or school contexts. The result suggests that interactions among neighborhood youth might be better conceptualized as reflecting a microsystem, as a primary socialization setting, than an exosystem as identified by the theory. Indeed, neighborhoods present unsupervised opportunities to smoke that are not possible in schools. Adolescents may be more aware of and influenced by smoking among neighborhood youth than by the larger group of school youth, as suggested by the school context modeling effect being substantially weaker than in the other contexts. Our

Table 3. Social context predictors of smoking from age 11 through age 17 years (*N* = 6,544)

	Model 1	Model 2	Model 3	Model 4	Model 5
			Mesosystem		
	Microsystem	Exosystem	Family × Peers	Family × School	Peers × School
	B (*SE*)	*B* (*SE*)	*B* (*SE*)	*B* (*SE*)	*B* (*SE*)
Family context					
Modeling	0.12 (0.02)***	0.12 (0.02)***	0.11 (0.02)***	0.09 (0.02)***	0.12 (0.02)***
Closeness	−0.03 (0.01)**	−0.03 (0.01)**	−0.03 (0.01)**	−0.03 (0.01)**	−0.03 (0.01)**
Social regulation	−0.02 (0.01)**	−0.02 (0.01)**	−0.02 (0.01)**	−0.02 (0.01)†	−0.02 (0.01)**
Strain	0.03 (0.00)***	0.03 (0.00)***	0.03 (0.00)***	0.02 (0.01)***	0.03 (0.00)***
Closeness × Model	−0.02 (0.01)*	−0.02 (0.01)*	−0.01 (0.01)*	−0.01 (0.01)†	−0.02 (0.01)*
Regulation × Model	−0.01 (0.00)**	−0.01 (0.00)**	−0.01 (0.00)*	−0.01 (0.00)*	−0.01 (0.00)**
Strain × Model	0.02 (0.00)***	0.02 (0.00)***	0.01 (0.00)***	0.01 (0.00)***	0.02 (0.00)***
Peer context					
Modeling	0.12 (0.02)***	0.12 (0.02)***	0.04 (0.05)	0.11 (0.02)***	0.10 (0.02)***
Closeness	−0.005 (0.00)†	−0.005 (0.00)†	−0.005 (0.00)†	−0.005 (0.00)†	−0.004 (0.00)
Social regulation	0.01 (0.00)**	0.02 (0.00)**	0.02 (0.00)**	0.02 (0.00)**	0.01 (0.00)**
Strain	0.10 (0.02)***	0.10 (0.02)***	0.10 (0.02)***	0.10 (0.02)**	0.08 (0.03)*
Closeness × Model	0.02 (0.01)**	0.02 (0.01)*	0.02 (0.01)*	0.02 (0.01)*	0.02 (0.01)**
Regulation × Model	0.02 (0.01)*	0.02 (0.01)*	0.02 (0.01)*	0.02 (0.01)*	0.02 (0.01)*
Strain × Model	0.00 (0.04)	0.00 (0.04)	0.00 (0.04)	0.00 (0.04)	−0.02 (0.04)
School context					
Modeling	0.52 (0.18)**	0.59 (0.18)**	0.57 (0.18)**	0.56 (0.20)**	0.51 (0.18)**
Closeness	0.01 (0.02)	0.00 (0.02)	0.00 (0.02)	−0.01 (0.02)	0.00 (0.02)
Social regulation	0.00 (0.05)	0.15 (0.05)**	0.14 (0.05)**	0.14 (0.05)*	0.14 (0.05)*
Strain	0.04 (0.06)	−0.01 (0.06)	0.00 (0.06)	0.02 (0.06)	0.01 (0.06)
Closeness × Model	−0.11 (0.09)	−0.05 (0.09)	−0.04 (0.08)	−0.03 (0.08)	−0.06 (0.09)
Regulation × Model	0.05 (0.28)	−0.64 (0.27)*	−0.63 (0.27)*	−0.54 (0.28)†	−0.57 (0.28)†
Strain × Model	−0.10 (0.23)	−0.05 (0.23)	−0.07 (0.23)	−0.20 (0.22)	−0.12 (0.23)
Neighborhood context					
Modeling	—	0.65 (0.03)***	0.65 (0.03)***	0.65 (0.03)***	0.65 (0.03)***
Closeness	—	−0.01 (0.02)	−0.01 (0.02)	−0.01 (0.02)	−0.01 (0.02)
Social regulation	—	0.01 (0.03)	0.00 (0.03)	0.01 (0.03)	0.01 (0.03)
Strain	—	−0.02 (0.05)	−0.02 (0.05)	−0.01 (0.05)	−0.02 (0.05)
Closeness × Model	—	−0.09 (0.14)	−0.09 (0.14)	−0.09 (0.14)	−0.09 (0.14)
Regulation × Model	—	0.01 (0.14)	0.02 (0.14)	0.00 (0.14)	0.01 (0.14)
Strain × Model	—	0.06 (0.30)	0.08 (0.30)	0.03 (0.31)	0.07 (0.31)
Family × Peer contexts					
Family closeness × Peer model	—	—	0.01 (0.01)	—	—
Family regulation × Peer model	—	—	−0.01 (0.01)	—	—
Family strain × Peer model	—	—	0.02 (0.01)*	—	—
Family model × Peer model	—	—	0.05 (0.01)***	—	—
Family × School contexts					
Family closeness × School model	—	—	—	−0.05 (0.05)	—
Family regulation × School model	—	—	—	−0.06 (0.04)	—
Family strain × School model	—	—	—	0.06 (0.02)*	—
Family model × School model	—	—	—	0.17 (0.03)***	—
Peer × School contexts					
Peer closeness × School model	—	—	—	—	−0.01 (0.02)
Peer regulation × School model	—	—	—	—	0.02 (0.03)
Peer strain × School model	—	—	—	—	0.12 (0.16)
Peer model × School model	—	—	—	—	0.14 (0.09)
F[a]	65.76***	0.37	9.67***	11.66***	1.04
BIC	37,912.74	37,192.76	37,144.33	37,136.12	37,203.05
−2 log likelihood	37,694.51	36,948.14	36,879.41	36,871.20	36,938.12

Note. Adolescents are nested in neighborhoods. All hierarchical growth models are computed on five imputed datasets and controlled for gender, race/ethnicity, family structure, parent education, and high school enrollment. BIC, Baysian Information Criterion.

[a]The *F* statistic tests the significance of the variables added in each model to the previous model. The microsystem model is compared with a model including only demographic variables.

†$p < .10$; *$p < .05$; **$p < .01$; *** $p < .001$.

findings suggest that neighborhoods as a primary source of role models for smoking deserve further empirical attention.

Although modeling effects were present in all four contexts, significant effects of the bond variables suggested by social control theory were present only in the peer and family contexts. These findings indicate that, at least for the social control variables we examined, these two microsystems are more important to youth smoking that the school and neighborhood contexts. The absence of school bond effects is notable given the theory's identification of the school as a microsystem. Our lack of findings contrasts with two other recent studies of school contextual effects on adolescent problem behaviors (Battistich & Hom, 1997; Henry et al., 2009), although neither of these studies focused specifically on cigarette smoking. Perhaps middle and high schools due to their larger size compared with elementary schools are less apt to function as a primary socialization context. Alternatively, our lack of findings may be due to the measures we used. Had we measured the adolescent's perceptions of the school environment, as is commonly done, rather than system-level variables representing the school as a whole, our results might have differed. The theory, however, stresses the importance of analyzing microsystems as systems.

The lack of association of any of the neighborhood social bond variables with youth smoking also was contrary to our hypotheses and expectations based on models of neighborhood collective efficacy (Sampson et al., 1997). Smoking might be perceived by neighborhood adults as less serious behavior than violence and delinquency and thus not subject to the same constraining influences.

In contrast, our findings provide support for one of the central tenets of the ecology of human development in the presence of interactions between settings, namely, the validity of the construction of mesosystems. We found interactive effects between the family and peer contexts and between the family and school contexts, although not between the peer and school contexts. Our results indicate that negative family characteristics exacerbate exposure to smoking among peers and classmates, although conventional family characteristics do not buffer such exposures. While not all interactions examined were significant, our results nevertheless suggest the need to consider the likelihood that effects stemming from any one context may be contingent on attributes of another context.

Hypothesized interactions between social learning and social control variables

Within the family and peer contexts, we found significant interactions between the social learning and social control variables; as hypothesized, the relationships were opposite each other. Overall, the findings suggest that in these two most fundamental developmental contexts, effects on adolescents of proximity to smokers cannot be estimated in absence of consideration of the nature of relations, and effects of social bonds cannot be described without reference to the smoking present in each context. But in addition, consideration of the specific context is required because the nature of the relationships vary.

In the family context, conventional bonds as evidenced by closeness and parental supervision buffered the effect of exposure to smoking by parents and siblings. Our findings are consistent with our expectation that strong conventional bonds in the family would promote prosocial behavior in adolescents by having a dampening effect on any tendency to imitate smoking by parents. Some studies also have reported buffering of parental smoking by attachment (Wilson et al., 2007) and parental control (Den Exter Blokland et al., 2007), although others have not found interactions (Chassin et al., 2005; White et al., 2000) or found a magnifying effect of attachment on parental smoking (Foshee & Bauman, 1992). Comparisons with other studies are limited, however, because of differences in smoking outcomes, the other variables in the model, and analytic techniques. Nonetheless, the literature suggests that consideration of other parenting processes, such as specific antitobacco socialization practices (e.g., Jackson & Henriksen, 1997; Otten, Engels, & van den Eijnden, 2008), may be needed to understand the interplay of factors in the family context relevant to youth smoking. Our findings also demonstrate, in contrast to common perceptions but consistent with other research (Bauman, Carver, & Gleiter, 2001), that family influences continue rather than wane from early to middle adolescence.

In the peer context, also as expected but opposite to the family, closeness to friends and peer social regulation (relationship closure) magnified the association between youth and friends' smoking. While few studies have examined interrelationships between peer smoking and other friendship attributes (Ennett, Faris, et al., 2008), our findings are consistent with those of Urberg et al. (2003) who found that effects of friends' smoking on adolescent smoking was more pronounced in higher quality friendships. Our findings also are consistent with developmental research, suggesting that friendship bonds serve to reinforce adolescent norms (Hartup, 1996).

While family strain (conflict) magnified the effect of parents and siblings' smoking as expected, peer strain (intransitivity) did not moderate the effect of friends' smoking but had a main effect. As expected, adolescents who belonged to higher proportions of intransitive triads, where friends of friends were not also friends, reported higher smoking. These adolescents may have been exposed to varying norms of behavior or expectations necessary for maintaining nonoverlapping friendships that negated moderation and engendered strain that led to smoking. These intransitivity findings are consistent with those of previous studies that show an association between smoking and social network isolation, another measure of friendship strain (Aloise-Young, Graham, & Hansen, 1994; Ennett & Bauman, 1993).

Whereas joint consideration of social learning and social control processes appears indicated in the family and peer contexts, our hypotheses about interactions between modeling and bond variable within schools and neighborhoods were not born out. Whether our lack of findings is due to diminished relevance of the nature of bonds in these large and less immediate settings or to the particular attributes we measured is unknown. Perhaps in these large settings, the adolescents' perceptions of contextual features become as important as the aggregated more objective measures we used.

Limitations

Our findings should be considered in light of methodological limitations. While our analysis of time-varying measures

demonstrated the contribution of social context characteristics to smoking averaged across all ages examined, we did not test differences at each age in the relationships between the social context variables and smoking. Furthermore, our statistical models, while based on longitudinal data, did not allow us to assess temporality of relationships. The models assessed the contemporaneous relationships between the time-varying social context measures and smoking at each timepoint assessed; the models did not assess whether the social context attributes at earlier ages predicted smoking at subsequent ages after controlling for prior smoking. Other statistical models are needed to conduct these analyses and are an essential consideration for future research. Note that the limitation concerning temporality precludes us from determining whether the relationship between adolescent smoking and their friends' smoking is due to the adolescent's selection of friends or to socialization by those friends. Prior research suggests that both selection and socialization are likely at work (Bauman & Ennett, 1996); indeed, the prediction from Bronfenbrenner's perspective would be that reciprocal processes exist between adolescents and peers.

Despite limitations, our study addresses challenges to contextual research in our use of theory to identify specific attributes of contexts and contextual interdependencies for examination. Our findings affirm Bronfenbrenner's social ecological perspective in demonstrating that adolescent smoking is socially conditioned behavior and that family, peer, school, and neighborhood social contexts are significantly implicated in adolescent smoking. Even so, the family and peer contexts were primarily implicated, with findings suggesting the need for consideration of interactive effects between social learning and social control variables within and between these contexts.

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Declaration of Interests

None declared.

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